Heart Health in Older Adults

Import of Heart Disease and Opportunities for Maintaining Cardiac Health

LINDA P. FRIED, MD, MPH, AND ROBERT L. MCNAMARA, MD, MHS, BALTIMORE, MARYLAND; GREGORY L. BURKE, MD, MS, WINSTON-SALEM, NORTH CAROLINA; AND DAVID S. SISCOVICK, MD, MPH, SEATTLE, WASHINGTON

Coronary heart disease remains the leading cause of morbidity and mortality in older adults,despite improved survival and declining mortality. This article describes the prevalence and impact of heart disease on people's lives, singly and in combination with other diseases. It then reviews current findings as to the risk factors for CHD in older adults and the underlying physiologic changes of aging plus pathophysiologic changes of hypertension and CHD in impairing the ability of older adults to respond to exercise and other stressors, and the effects of exercise training in attenuating the adverse cardiovascular changes of aging. This information provides a basis for considering opportunities for prevention of heart disease and maximizing heart function. The article concludes by describing the known contribution of preventive measures to declines in heart disease in older adults.

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Coronary heart disease remains the leading cause of morbidity and mortality in older adults, even though survival has improved[1,2] and its incidence in the United States has declined over the past few decades.[3,4] The acute death rate from coronary disease has decreased substantially more than the chronic death rate (4.0% annually versus 2.5%),[3,5,6] leading to conclusions that survival rates in patients with heart disease are improving. Consistent with this, prevalence rates are rising among older adults, resulting in an increase in the absolute number of older adults with coronary heart disease.[3,7] More older adults are living—and living longer—with chronic heart disease. We will examine here the prevalence of and risk factors for cardiovascular disease in older adults, the physiology of aging and the effects of exercise, so as to consider opportunities for maintaining cardiovascular health.

The import of coronary heart disease in older adults is reflected, in part, by the prevalence rates. In the Cardiovascular Health Study (CHS), a population-based study of 5,201 men and women aged 65 years and older in four US communities,[8] the prevalence of heart disease increased with age (5-year age groups, from 65 to 69 to 85 and older) and varied by sex. Myocardial infarction (MI) was found in 11% to 18% of men and 4% to 9% of women older than 65, varying with age group; angina pectoris was found in 15% to 17% of men and in 8% to 13% of women; and congestive heart failure was present in 1% to 3% of both men and women between 65 and 84 years.[9] Prevalences of MI and congestive heart failure were higher in black men and women than in whites, whereas the rates for angina pectoris were slightly lower.[10] In addition, the prevalence of unreported, and possibly silent, MIs was high; 23% of men and 38% of women 65 and older with electrocardiographic evidence of old MI did not have a known history of infarction.[9] In the same population, 13% of men and 9% of women had ischemic episodes on 24-hour ambulatory electrocardiograms, including 9.6% of participants without any history of MI and no major or minor resting electrocardiographic abnormalities.[11] Overall, 28.9% of men and 19.3% of women 65 and older had heart disease.[12] In comparison, the Women's Health and Aging Study, which evaluated the third most

From the Johns Hopkins University School of Medicine (Drs Fried and McNamara), Bowman Gray School of Medicine at Wake Forest University/Baptist Hospital and Medical Center (Dr Burke), and the University of Washington School of Medicine (Dr Siscovick).

Reprint requests to Linda P. Fried, MD, MPH, 2024 E Monument St, Ste 2-600, Baltimore, MD 21205.

ABBREVIATIONS USED IN TEXT

A-$\dot{V}O_2$ = arteriovenous oxygen
CHS = Cardiovascular Health Study
MI = myocardial infarction
$\dot{V}O_2max$ = maximum oxygen consumption

disabled women aged 65 years and older living in the community, 20.9% reported a history of MI, 20.8% a history of angina pectoris, and 8.3% a history of congestive heart failure.[13]

Subclinical cardiovascular disease is also prevalent in older adults. A summary index of subclinical disease was created in the CHS, based on measurements of several vascular beds using electrocardiographic and echocardiographic abnormalities and common carotid artery wall thicknesses as measured by ultrasonography, ankle-brachial blood pressure, and positive response to the Rose Questionnaire for angina. Using this index, 36.1% of women and 38.7% of men in the CHS had subclinical disease only. The prevalence of subclinical disease increased with age, so that only 12.6% of those aged 85 years and older had neither clinical nor subclinical cardiovascular disease.[14]

The import of coronary heart disease in older adults is also expressed by its effects on individual patients. Heart disease is among the major causes of disability that we associate with aging, whether evaluated by aggregate associations or through individuals. Regarding the latter, participants in the CHS cited heart disease, along with lung disease, as the most frequent cause of difficulty in tasks requiring the ability to perform physical work or exercise tolerance, such as walking half a mile, doing heavy housework, walking up ten steps, and lifting or carrying 4.5 kg (10 lb).[12] Shortness of breath was the symptom most associated with difficulty in these same tasks. In analytic approaches used to understand the association of heart disease with function, angina pectoris has been shown in the Framingham Study to be predictive of disability; angina pectoris was more predictive than MI or coronary artery insufficiency.[15] In the CHS, cross-sectional analysis showed that angina pectoris requiring nitroglycerin use, MI, congestive heart failure, shortness of breath, and left ventricular systolic dysfunction by echocardiography were each independently associated with difficulty in performing the tasks of daily life, ranging from mobility and exercise tolerance–demanding tasks to basic self-care tasks.[16] In addition, there is early evidence from the CHS cohort that subclinical cardiac functional impairments, as measured by echocardiography, may be associated with disability as well (L.P.F., D.S.S., J. M. Gardin, H. S. Klopfenstein, J. A. Robbins, and P. J. Savage for the Cardiovascular Health Study Group: "Association of Subclinical Cardiac Impairments With Physical Disability in Older Adults," (1997). These data all indicate that heart disease has substantial effects on the well-being and independence of older adults. A summary expression of this has been provided by Kosorok and associates, who estimated that 4 of the 31 restricted-activity days per person per year reported by older adults were due to heart disease.[17] Another calculation, of population attributable risk, indicates that 9% of disability and dependency in older adults is caused by heart disease.[18] Given this perspective, the results of the effective prevention and treatment of cardiac disease will include preventing the disability and loss of independence associated with aging.

Heart disease also interacts with other diseases in causing adverse consequences. This has been best studied in terms of the synergistic effects of heart disease and arthritis. In a study by Ettinger and colleagues, those with heart disease but no arthritis had a 2.3-fold increased risk of having difficulty in ambulation over those with neither disease. When arthritis was also present, the relative risk for both heart disease and knee osteoarthritis was 13.6.[19] Clinical observation suggests that a good proportion of this interaction may lie in the decreased efficiency of ambulation and increased energy demands and stress on the heart in the presence of arthritis of the lower extremities.[20] Heart disease and arthritis commonly coexist, and particularly so in disabled persons. For example, in the Women's Health and Aging Study's population of moderately to severely disabled older women, 10% reported both heart disease and arthritis (L.P.F., K. Bandeen-Roche, J. D. Kasper, and J. M. Guralnik: "Association of Comorbidity With Disability in Older Women: The Women's Health and Aging Study," 1997). Overall, 50% of older adults have two or more chronic diseases. The joint effects of heart disease with other diseases need to be considered in treatment goals, as do the secondary effects of medications for heart disease on other conditions.

Thus, both clinical and subclinical cardiac diseases are prevalent in older adults. Changing rates, as described earlier, suggest that these rates can be modified.

Risk Factors for Cardiovascular Disease

Factors associated with the incidence of MI in adults older than 65 years also were assessed in the CHS.[21] Over about a five-year follow-up period, incidence rates of MI were higher in older participants and in men (21 per 1,000 person-years) than in women (7 per 1,000 person-years).[22] Predictors of incident MI included elevated systolic blood pressure, cigarette smoking, and elevated serum glucose levels. Of interest, in this cohort of older adults, serum lipid or lipoprotein measurements were not significant predictors of incident MI. A number of measures of subclinical disease, including the intimalmedial thickness of the internal carotid artery, decreased cardiac ejection fraction, and a low ankle-arm index, were significant predictors of incident MI after adjustment for age and sex. Based on these results, modifiable risk factors such as blood pressure, smoking, and serum glucose levels are associated with an increased risk of MI, and interventions on these factors may facilitate the primary prevention of this disease in older adults.

A number of risk factors are shared between incident MI and stroke in older adults. Factors associated with incident stroke rates in older adults over about a three- to four-year follow-up period were assessed in the CHS cohort.[23] The stroke incidence was similar in men and women and increased with age.[24] Incident stroke in these older adults was associated with diabetes mellitus, elevated systolic blood pressure, increased left ventricular mass or wall motion abnormalities, carotid artery stenosis, and atrial fibrillation. Of interest, aspirin users free of coronary heart disease, atrial fibrillation, transient ischemic attacks, or claudication at rest had a 52% increase in stroke incidence even after adjustment for other risk factors. The stroke incidence also was associated with elevated levels of modifiable risk factors and with subclinical disease measures. Perhaps more important is the fact that interventions that reduce the burden of incident MI in older adults—elevated systolic blood pressure, possibly glycemic status, and the like—would also be expected to favorably affect stroke incidence rates.

In this study, prevalent subclinical cardiovascular disease was associated with a number of risk factors also associated with clinical disease: age, systolic blood pressure, fasting glucose levels, high blood pressure, and smoking in both women and men, and, in addition in women, diastolic blood pressure, low-density lipoprotein–cholesterol levels, high-density lipoprotein–cholesterol levels, and leukocyte count. In addition, isolated systolic hypertension was associated with subclinical cardiac diastolic function and increased intimalmedial thickness of the carotid artery.[25]

In particular, carotid artery ultrasonography has been used to noninvasively assess factors associated with atherosclerosis in numerous population-based studies.[26,27] In the Atherosclerosis Risk in Communities (ARIC) study of middle-aged adults, a number of factors were associated with increased carotid artery wall thickness, including age, hypertension, active and passive cigarette smoking, serum lipid or lipoprotein levels, diabetes mellitus, dietary antioxidant and fat intake, and hemostatic factors.[28–36] In the CHS study of older adults, factors associated with atherosclerosis include cigarette smoking, ankle-arm index, hormone replacement therapy (women), hemostatic factors, diabetes mellitus and glucose intolerance, hypertension, and left ventricular hypertrophy.[37–41] These data document the importance of behavioral and clinical factors on the burden of atherosclerosis in middle-aged and older adults.

Several recent reports highlight the opportunities for maintaining cardiac health and the effectiveness of prevention and treatment. First, in the CHS, the presence of subclinical cardiovascular disease identified older adults who are at high risk of progressing to clinical disease.[42] Overall, over a three-year follow-up period, those with subclinical disease had a twofold higher risk of coronary heart disease and mortality than those without subclinical disease. In women, 43% of incident coronary heart disease was due to subclinical disease; in men, the proportion was 37%. These data suggest that the primary prevention of subclinical cardiovascular disease is likely to have a substantial impact on rates of clinical disease. Given, however, that in CHS, almost half of older adults had subclinical disease at the baseline evaluation, effective secondary prevention of clinical cardiovascular disease is also critical. This is accomplished through targeting those with subclinical disease for interventions to minimize factors that precipitate transitions to clinical disease.

Cardiovascular Changes With Aging

Clear differences exist in the cardiovascular systems of young and older people. How much these differences can be attributed to the pathophysiologic effects of disease and how much to the physiologic effects of aging are less clear. The increased prevalence of disease with age, particularly coronary artery disease and hypertension, complicates the distinction. Changes in the heart and vasculature associated with aging, however, may interact with the effects of superimposed disease, possibly altering the effectiveness of therapy. Prevention and the treatment of disease have contributed substantially to the decreasing rate of cardiovascular mortality over the past three decades.[3] In addition, delays in the physiologic effects of aging may also have contributed. We summarize some relevant concepts derived from studies that sought to isolate the physiologic effects of aging from the pathologic effects of disease, background information essential to maximizing prevention and therapy.

Arterial vessel walls undergo major structural changes with age, including increased collagen deposition and hypertrophy of the smooth muscle cells, that in turn lead to decreased vascular elasticity.[43,44] These structural changes are compounded by a decrease in β-adrenergic receptor reactivity,[45,46] leading to a decreased capacity for vasodilatation. The resultant increase in systolic blood pressure, and thus afterload, with age has been repeatedly shown.[46–49] Less known, but potentially as detrimental, is an increase in the pulse-wave velocity.[47,48] In normal physiology, a pressure wave is referred from the aorta back to the heart during diastole, aiding coronary perfusion. The age-associated increase in pulse-wave velocity produces the referred pressure wave during systole, further increasing afterload.

Together, the increases in systolic pressure and pulse-wave velocity contribute to the age-associated structural and functional changes in the myocardium, which primarily affect diastolic function. The increased afterload causes connective tissue deposition and increased myocyte size,[43,50] which leads to left ventricular wall thickening[51,52] and decreased left ventricular compliance. Decreased β-adrenergic activity in the myocardium also impairs relaxation.[45,46] As a result, diastolic filling abnormalities can be seen on echocardiography. The peak velocity of the rapid early diastolic filling (the "E" wave on M-mode echocardiography) decreases, and the late component (the "A" wave) increases.[53]

Systolic function is less affected by the aging process. The resting ejection fraction does not appear to

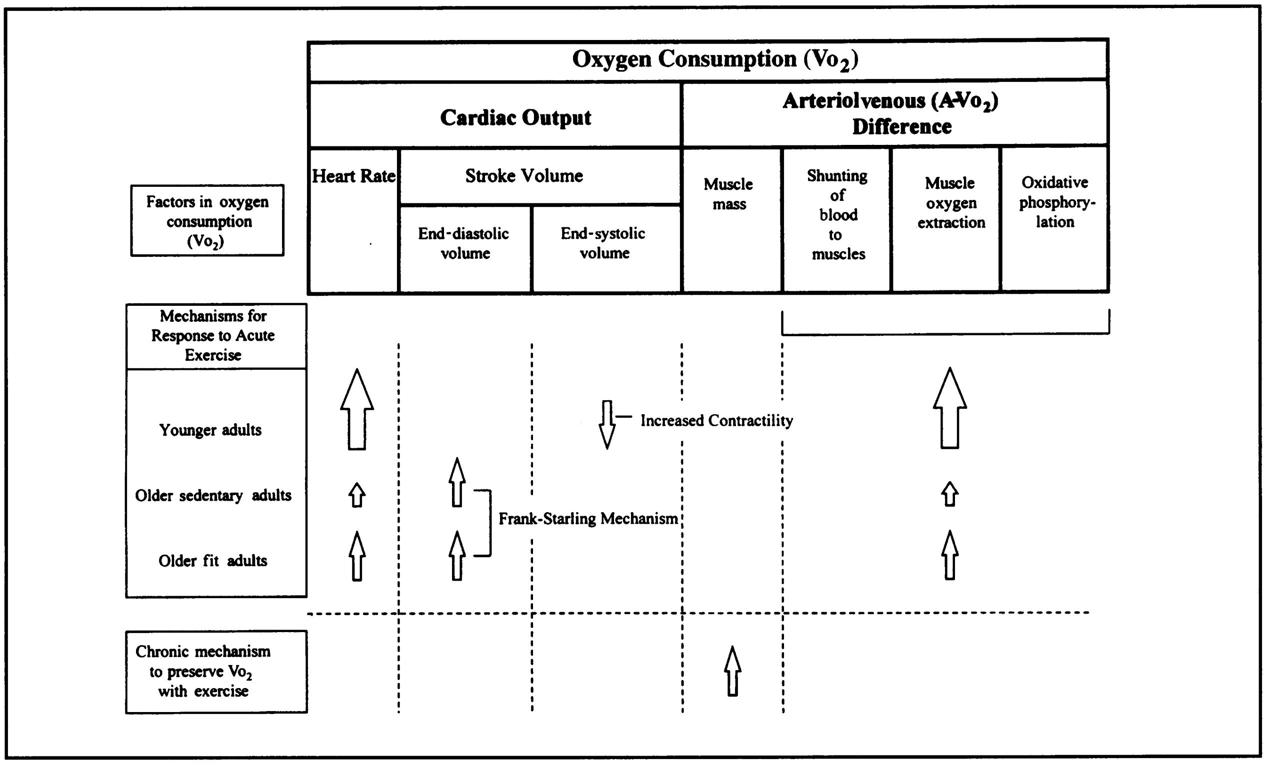

Figure 1.—Physiologic factors that make up oxygen consumption are shown. Different age and fitness groups respond to exercise by relative magnitudes of mechanisms (as shown by the different-sized arrows), and an important long-term mechanism can preserve maximum oxygen consumption.

change with age.[49,54–56] The resting heart rate and cardiac output remain relatively constant because of an increase in the level of catecholamines[45] to offset the decreased β-adrenergic receptor activity. Overall, the resting heart adapts well to the structural and functional changes associated with aging.

Effect of Exercise on the Physiology of Aging

Age-associated changes in the cardiovascular system, however, become more apparent during exercise. Most notably, the maximum oxygen consumption ($\dot{V}o_2max$) with exercise decreases substantially. Both components of the $\dot{V}o_2max$, cardiac output and arteriovenous oxygen (A-$\dot{V}o_2$) difference, are affected (Figure 1). In normal, younger hearts, cardiac output during exercise is primarily augmented by increasing heart rate and increasing oxygen extraction, with some increase in contractility (decreased end-systolic volume). In older adults, the maximum heart rate achieved with exercise is substantially attenuated.[45,46,49,56,57]

An overall exercise-induced increase in the stroke volume, the other component of cardiac output, appears to be preserved with aging.[46,54,56] Older hearts, however, increase their stroke volume using a different physiology than younger hearts. Younger adults, particularly men, rely on increasing contractility, producing a decreased end-systolic volume. Older adults instead increase their end-diastolic volume through the Frank-Starling mechanism to increase cardiac output during exercise.[46,54,56] Interestingly, younger women may also rely more on the Frank-Starling mechanism than do younger men,[55,58] though conflicting findings have been reported.[59] More research is needed to define gender differences in the physiology of aging.

The $\dot{V}o_2max$ depends not only on cardiac output but on the A-$\dot{V}o_2$ difference. The A-$\dot{V}o_2$ difference increases with exercise in all ages, but less so in older adults.[45] Decreased muscle mass in older adults, compared with younger adults, explains part of this attenuation of oxygen consumption. The abilities to shunt blood to muscle and of the muscle to extract the oxygen and the rate of oxygen phosphorylation are other mechanisms that younger adults use to increase oxygen consumption with exercise that are attenuated in older adults.[45,59,60]

In summary, changes associated with aging primarily affect arterial elasticity, ventricular compliance, and β-adrenergic receptor activity. Although these changes are well tolerated at rest, in the presence of stressors on the heart, such as exercise, older adults have an impaired ability to increase heart rate and contractility, leading to a reliance on the Frank-Starling mechanism to augment cardiac output. Oxygen extraction is also attenuated in older adults, primarily through decreased muscle mass. Superimposed on these physiologic changes of aging, diseases such as hypertension and coronary artery dis-

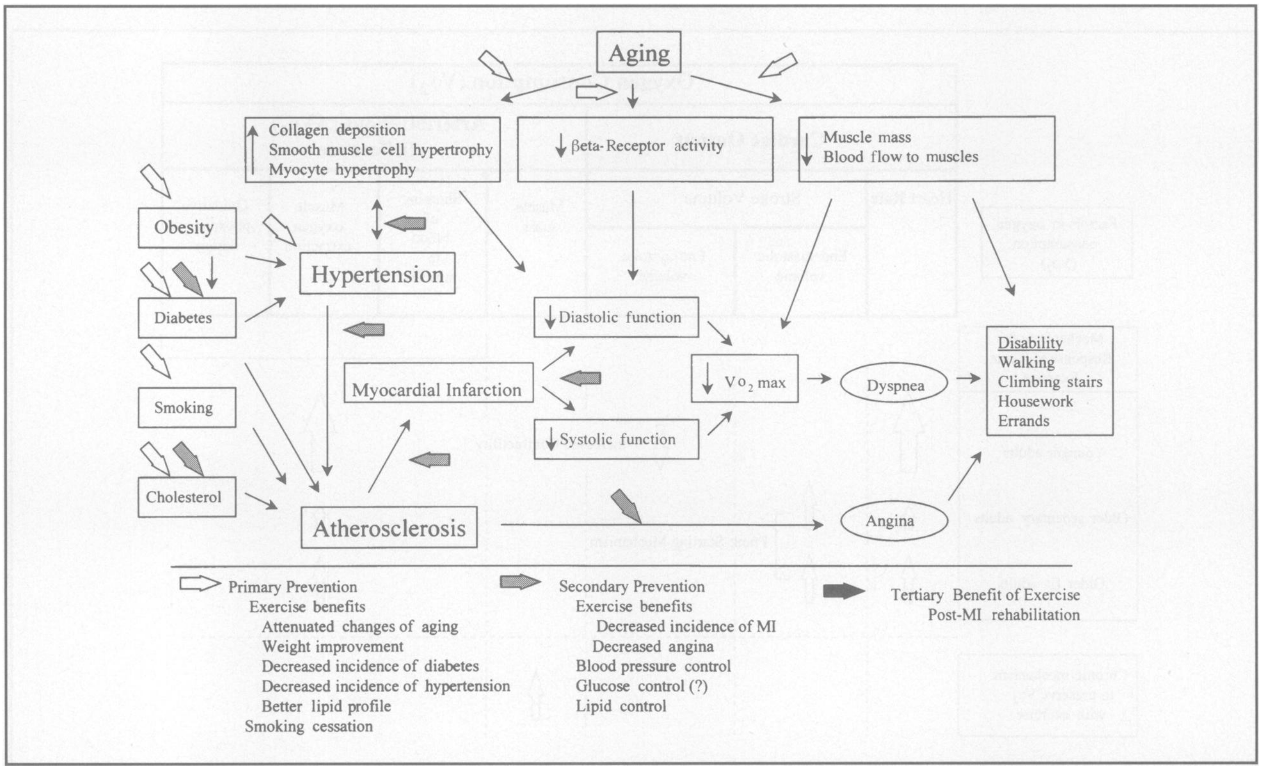

Figure 2.—The schematic shows age and disease effects on the development of disability and possible target areas for intervention. The thick arrows in the diagram correspond to the levels of prevention listed below the diagram. MI = myocardial infarction, VO_2max = maximum oxygen consumption

ease further impair the ability of older adults to respond to exercise and other stressors.

Effects of Exercise Training

As discussed earlier, clear differences in the way older adults respond to exercise can be identified. Is prevention possible? An exercise training program can attenuate some of the adverse changes in the cardiovascular system with aging (Figure 1). In one study, a decline with age in the $\dot{V}o_2$max was 50% greater in sedentary men and women than in their exercise-trained counterparts.[57] Exercise training was defined as 30 minutes of strenuous exercise three to four times a week for several years. In fact, the $\dot{V}o_2$max was higher in exercise-trained elderly men and women than in sedentary men and women 30 to 40 years younger. Age-related effects on maximum heart rate and oxygen extraction were attenuated in the trained subjects. In another study, exercise training was shown to improve the $\dot{V}o_2$max by peripheral adaptations without increasing end-diastolic volumes or contractility.[60]

In addition to increasing exercise capacity directly, exercise training has been shown to affect positively many age-related changes in cardiovascular risk factors and physiology at rest, including body fat, blood pressure, cholesterol level, and muscle mass.[61] The sedentary lifestyle that many older adults have, whether they are community-dwelling or residing in assisted-living facilities, may exacerbate losses of muscle function. A high-intensity strength program, even in institutionalized people, has been shown to result in increased strength and functional performance.[62]

Opportunities for Maximizing Cardiac Health

Cardiovascular disease remains the leading cause of morbidity and mortality in older adults. The prevalence of heart disease, both clinical and subclinical, increases with age. Serious comorbid diseases in older adults interact with heart disease to cause greater morbidity and mortality. The physiologic effects of aging occur in conjunction with the adverse effects of disease on the cardiovascular system and in combination may lead to disability in older adults (Figure 2). Modifying risk factors such as stopping smoking, controlling blood pressure,[63,64] and managing glucose levels are keystones of prevention. The benefits of lipid management in older adults remain controversial.[65,66] Exercise training appears clearly beneficial in augmenting primary and secondary prevention, including directly decreasing the incidence of MI[67,68] and lessening anginal symptoms.[69] Exercise is also an indispensable component of post-MI rehabilitation.[69,70]

In the absence of incapacitating cardiovascular disease, dementia, or gait disturbances, recommendations for exercise in older adults are not substantially different from those for younger people,[71] including 5 to 10 minutes of warm-up, 20 to 40 minutes of aerobic exercise at 60% to 85% maximum heart rate, and 5 to 10 minutes of cool down, three to five days per week. Starting slowly and progressing gradually permit an appropriate exercise program.

Both primary and secondary prevention of coronary heart disease appear to be effective on a population basis. Hunink and co-workers recently reported that for the period 1980 to 1990, primary prevention explained 25% of the decline in the mortality of coronary heart disease through a decrease in its incidence.[3] Moreover, 29% of the decline was explained by a secondary reduction in risk factors in patients with coronary artery disease and 43% by other improvements in the treatment of patients with coronary heart disease. These findings indicate both success to date from clinical approaches to prevention and treatment and optimism for the future in maintaining cardiovascular health. Approaches designed specifically for older adults should yield even higher rates of improvement. Thus, primary and secondary prevention through risk factor modification and exercise are keys to successful cardiovascular aging.

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